Polycystic ovary syndrome: A review for dermatologists

Part I. Diagnosis and manifestations

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Polycystic ovary syndrome (PCOS) is a common endocrine disorder among women who are of reproductive age. The pathogenesis involves several associated hormonal pathways that culminate in metabolic, reproductive, and cardiovascular effects. The hallmark features of hyperandrogenism and hyperinsulinemia have systemic long-term implications. Dermatologists frequently evaluate and manage the cutaneous manifestations of PCOS (ie, acanthosis nigricans, hirsutism, acne, and alopecia), and therefore play a key role in its diagnosis and management. In part I of this continuing medical education article, we review the definition, etiology, pathogenesis, and clinical features of PCOS. (J Am Acad Dermatol 2014;71:847.e1-10.)

Key words: acanthosis nigricans; acne; anovulation; hirsutism; hyperandrogenism; insulin resistance; polycystic ovary syndrome.

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age. This pervasive disorder of unknown etiology is characterized by 3 fundamental features: hyperandrogenism, chronic anovulation, and ultrasonographic evidence of polycystic ovaries. Women with PCOS are at risk for multisystemic consequences, including type 2 diabetes mellitus, cardiovascular disease, endometrial cancer, obstructive sleep apnea, nonalcoholic steatohepatitis, and psychiatric disorders. Clinicians involved in the care of women with PCOS should understand the potential health risks for these patients. Dermatologists are in a unique position to recognize the clinical manifestations of hyperandrogenism and insulin resistance and play an important role in the diagnosis and management of women with PCOS.

Abbreviations used:

BMI:	body mass index
DHEA:	dehydroepiandrosterone
DHEAS:	dehydroepiandrosterone sulfate
FSH:	follicle-stimulating hormone
GnRH:	gonadotropin-releasing hormone
IGF-1:	insulin-like growth factor 1
LH:	luteinizing hormone
OSA:	obstructive sleep apnea
PCOS:	polycystic ovary syndrome
SHBG:	sex-hormone binding globulin

DEFINITION

Key points

- **Polycystic ovary syndrome is a common endocrine disorder that affects up to 8% of women who are of reproductive age**
- **The 2003 Rotterdam criteria requires 2 out of 3 clinical indications to make the diagnosis of polycystic ovary syndrome, including oligo- or anovulation, clinical and/or biochemical signs of hyperandrogenism, and echographic polycystic ovaries**
- **Polycystic ovary syndrome is a diagnosis of exclusion; other etiologies of hyperandrogenism and anovulation must be ruled out**
- **The etiology remains unknown, but genetics along with early androgen exposure likely play a role**

In 1935, Drs Irving Stein and Michael Leventhal described a phenomenon in which 7 women had anovulation and polycystic ovaries discovered during surgery.[1] The condition was called Stein–Leventhal syndrome and was later renamed polycystic ovary syndrome (PCOS) to represent the unique morphology of the ovaries. Since its initial description, 2 main definitions of PCOS have emerged. The 1990 National Institutes of Health (NIH) definition requires the presence of oligo- or anovulation and biochemical or clinical signs of hyperandrogenism. Alternatively, the 2003 Rotterdam criteria broadens this definition and requires the presence of 2 out of 3 of the following clinical indications: oligo- or anovulation, biochemical or clinical signs of hyperandrogenism, and echographic polycystic ovaries (Table I).[2] Importantly, both definitions require the exclusion of other conditions that result in anovulation and hyperandrogenism, such as congenital adrenal hyperplasia, Cushing syndrome, and androgen-secreting tumors. These conditions can be excluded upon the evaluation of symptoms and relevant laboratory studies (Table II). The Rotterdam criteria

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Table I. 1990 National Institutes of Health criteria and 2003 Rotterdam criteria for the diagnosis of polycystic ovary syndrome

National Institutes of Health criteria (requires all 3)
1. Chronic anovulation
2. Clinical and/or biochemical signs of hyperandrogenism
3. Exclusion of other causes of hyperandrogenism and anovulation, such as Cushing syndrome, congenital adrenal hyperplasia, and androgen-secreting tumors
Rotterdam criteria (requires 2 out of 3)
1. Oligo- or anovulation
2. Clinical and/or biochemical signs of hyperandrogenism
3. Echogenic evidence of polycystic ovaries and exclusion of other causes of hyperandrogenism and anovulation, such as Cushing syndrome, congenital adrenal hyperplasia, and androgen-secreting tumors

identify 4 phenotypes of PCOS, which illustrates the variable clinical features of irregular menses, hyperandrogenism, polycystic ovaries, and insulin resistance (Table III). The prevalence of PCOS among women who are of reproductive age has been estimated to be 6.5% to 8%.[3] However, the prevalence varies depending on the diagnostic criteria used. Given the broader definition described in the Rotterdam criteria, the prevalence of PCOS has subsequently been noted to range from 15% to 18%.[4-6]

While the etiology of PCOS is unknown, it is theorized that gestational environment and steroid exposure likely play a role. Early exposure to androgen excess in utero or during the neonatal period is associated with the development of the PCOS phenotype later in life[7,8]; this was shown in several primate and nonprimate animal studies.[7-9] Rhesus monkeys that were exposed to prenatal testosterone later developed higher basal androgen levels and an exaggerated androgen response when stimulated.[8] This was further supported by another study on female lambs that were exposed to intrauterine testosterone.[9] Those lambs subsequently developed the PCOS phenotype during adolescence and had enlarged ovaries, irregular menstrual cycles, and hyperandrogenism. Studies on humans with congenital virilizing tumors have shown continued metabolic and reproductive abnormalities similar to the PCOS phenotype, even after treatment.[7] These findings suggest that the hypothalamic-pituitary-gonadal axis is programmed by early androgen exposure. In addition to environmental factors, PCOS also has a genetic basis and is associated with several candidate genes for insulin resistance and androgen production (eg, cytochrome P450c17, cytochrome P450c11a, and insulin receptor substrate 1), supporting the evidence of strong heritability of PCOS in families.[10-12]

PATHOGENESIS

Key points

- **Patients with polycystic ovary syndrome have an increased pulsatility of gonadotropin-releasing hormone, which results in a preferential secretion of luteinizing hormone**
- **The hormonal pathways of polycystic ovary syndrome involve the interplay among androgens, insulin, luteinizing hormone, and estrogen, leading to broad metabolic and reproductive sequelae**

PCOS is a complex disorder with several aberrant hormonal pathways resulting in reproductive and metabolic abnormalities. While the pathogenesis is not completely understood, several key hormonal pathways likely contribute. In PCOS, the hypothalamus secretes gonadotropin-releasing hormone (GnRH) at an increased pulse frequency.[13] This increased GnRH frequency is either caused by an inherent defect in the GnRH pulse generator or to lower progesterone levels among women with PCOS. Progesterone slows the GnRH pulse generator, which explains why low levels of progesterone could increase GnRH pulsatility.[14] The net increased frequency of GnRH pulsation stimulates the anterior pituitary gland to preferentially secrete luteinizing hormone (LH) over follicle-stimulating hormone (FSH).[15] LH stimulates the ovarian theca cells to synthesize androstenedione. Androstenedione can either be converted to testosterone or it can be aromatized in the nearby ovarian granulosa cell and converted into estrogen via aromatase. While the theca cell is stimulated by LH, the granulosa cell is stimulated by FSH. In this setting of preferential LH secretion, the net ovarian hormonal production is an increased amount of androgen. Androgens have numerous local and systemic effects. They act locally to arrest ovarian follicular development, explaining the numerous immature follicles seen on ultrasound.[15] Androgens also have systemic effects on the development of hirsutism, acne, and central obesity.

Androgens are ultimately converted to estrogen by the peripheral adipose tissue, increasing net estrogen production. Estrogen stimulates proliferation and differentiation of the endometrium which, when unopposed by progesterone, can increase the risk for endometrial hyperplasia and tumorigenesis. Estrogen also inhibits the anterior pituitary gland

Table II. Differential diagnosis of polycystic ovary syndrome

Differential diagnosis	Clinical features	Laboratory evaluation
Pregnancy	Amenorrhea	Elevated serum or urine hCG
Premature ovarian failure	Amenorrhea	Elevated follicle-stimulating hormone, elevated LH, and low-normal estradiol levels
Hypothyroidism	Amenorrhea, fatigue, cold intolerance, constipation, and weight gain	Elevated thyroid-stimulating hormone and low thyroxine levels
Hyperprolactinemia	Amenorrhea and galactorrhea	Elevated prolactin level
Late-onset congenital adrenal hyperplasia	Hyperandrogenism and amenorrhea	Elevated day 5 morning level of 17-hydroxyprogesterone
Virilizing ovarian/adrenal tumor	Amenorrhea, hyperandrogenism, clitoromegaly, deepening of voice, increased muscle mass, and rapidly progressive hirsutism or alopecia	Total testosterone >200 ng/dL, DHEAS >700 μg/dL, and elevated androstenedione
Cushing syndrome	Hyperandrogenism, amenorrhea, hypertension, abdominal striae, truncal obesity, facial plethora, glucose intolerance, pedal edema, and easy bruisability	Elevated 24-hr urine free cortisol level, unsuppressed morning serum cortisol during the low-dose dexamethasone suppression test, and elevated midnight salivary cortisol

DHEAS, Dehydroepiandrosterone sulfate; *hCG*, human chorionic gonadotropin; *LH*, luteinizing hormone.

Table III. Polycystic ovary syndrome phenotypes based on the 2003 Rotterdam criteria[2]

Phenotype	Prevalence	Clinical features
Severe PCOS	61%	Irregular menses, polycystic ovaries, hyperandrogenemia, and hyperinsulinemia
Hyperandrogenism and chronic anovulation	7%	Irregular menses, normal ovaries, hyperandrogenemia, and hyperinsulinemia
Ovulatory PCOS	16%	Normal menses, polycystic ovaries, hyperandrogenemia, and hyperinsulinemia
Mild PCOS	16%	Irregular menses, polycystic ovaries, mildly raised androgen levels, and normal insulin levels

PCOS, Polycystic ovary syndrome.

from secreting FSH, which further contributes to preferential LH secretion. Insulin is another hormone involved in the pathogenesis of PCOS. Similar to LH, insulin stimulates the ovarian theca cell to secrete androgens. Insulin also inhibits hepatic production of sex hormone–binding globulin (SHBG), thereby elevating free testosterone. The net result is an increase in androgen levels. Finally, obesity plays an important role in these hormonal pathways by engendering insulin resistance, further stimulating the net production of androgens. Androgen excess contributes toward abdominal obesity, which subsequently propagates the cycle. Weight loss has been shown to effectively halt this cycle, restoring ovulation and decreasing insulin and testosterone levels among women with PCOS.[16-18]

CLINICAL FEATURES

Key points

- **Hyperandrogenism, oligo- or anovulation, and polycystic ovaries are the hallmark clinical features of polycystic ovary syndrome; other important features include insulin resistance, obesity, cardiovascular disease, obstructive sleep apnea, nonalcoholic steatohepatitis, and psychiatric disease**
- **Cutaneous manifestations of polycystic ovary syndrome include signs of insulin resistance, such as acanthosis nigricans, and signs of hyperandrogenism, such as hirsutism, acne, and hair loss**
- **Chronic anovulation predisposes patients to infertility and endometrial cancer**
- **Polycystic ovaries are defined by the presence of ≥12 2- to 9-mm diameter follicles in each ovary and/or increased ovarian volume (defined as >10 mL)**

PCOS has variable clinical manifestations. The 3 distinguishing features include hyperandrogenism, chronic anovulation, and ultrasonographic evidence of polycystic ovaries. Other important features evident among this population include insulin

Table IV. Multisystem effects of polycystic ovary syndrome

System	Manifestations
Endocrine	Type 2 diabetes mellitus, amenorrhea, and hyperandrogenism
Reproductive	Infertility and endometrial hyperplasia/cancer
Cardiovascular	Coronary artery disease, dyslipidemia, and hypertension
Dermatologic	Hirsutism, acne, alopecia, and acanthosis nigricans
Gastrointestinal	Nonalcoholic steatohepatitis
Pulmonary	Obstructive sleep apnea
Psychiatric	Depression and anxiety

resistance, cardiovascular disease, obstructive sleep apnea, nonalcoholic steatohepatitis, and psychiatric manifestations (Table IV).

Hyperandrogenism

Hyperandrogenism is one of the most important diagnostic features of PCOS and the most relevant to the role of dermatologists in diagnosis and management of the disorder. Clinical signs include hirsutism, acne, seborrhea, and less commonly hair loss. Any of these signs in addition to the presence of irregular menses should prompt consideration of the diagnosis of PCOS. An initial laboratory work-up includes serum total and free testosterone (calculated is most accurate), SHBG, dehydroepiandrosterone sulfate (DHEAS), prolactin, and a pelvic ultrasound. Other causes of amenorrhea and hyperandrogenism can be ruled out with laboratory tests (Table II). If there is high clinical suspicion for PCOS or another endocrinopathy, referral to an endocrinologist should be considered. Up to two-thirds of women with PCOS will have elevated total testosterone levels, which are associated with greater metabolic and reproductive morbidity.[19] Elevations in free testosterone, however, are felt to be a more sensitive marker of hyperandrogenemia.[2]

Notably, signs of virilization, such as deepening of the voice, increased muscle mass, and rapidly progressive hirsutism, are rare manifestations of PCOS and should prompt an evaluation for underlying androgen secreting tumors. Signs of androgen excess are most often evident during puberty, but can occur after puberty, especially in the setting of weight gain.

Hirsutism, defined as excessive terminal body hair in a male distribution, often suggests underlying hyperandrogenism. It is frequently seen on the upper lip, chin, areola, chest, back, and lower abdomen (Fig 1). Up to 60% of women with PCOS have hirsutism.[20,21] Women with hirsutism have increased follicularly based 5α-reductase activity, which is locally stimulated by androgens, insulin, and insulin-like growth factor.[22,23] Increased levels of 5α-reductase fosters the conversion of testosterone to dihydrotestosterone, which stimulates hair growth. The degree of hirsutism varies depending on ethnicity; women from South Asia tend to have a higher prevalence while women from Japan have a lower prevalence.[24-26] Hair follicles appear to have varying sensitivities among different ethnicities, explaining this disparity.[22,25]

Acne is another common manifestation of hyperandrogenism among women with PCOS. Compared with normal pubertal acne, women with PCOS have predominantly inflammatory lesions on the lower face, neck, chest, and upper aspect of the back. Women with moderate to severe acne should be investigated for PCOS, because 19% to 37% of patients with moderate to severe acne meet the criteria for this disorder.[27,28] Acne that originates or persists into adulthood and that is refractory to conventional therapies should raise suspicion for underlying PCOS. Ovarian and adrenal androgens, such as androstenedione, testosterone, dehydroepiandrosterone (DHEA), and DHEAS stimulate comedone production by binding to androgen receptors on the pilosebaceous unit, thereby increasing sebaceous gland size, activating sebum production and causing abnormal follicular epithelial cell keratinization.[22,29] Sebum production leading to *Propionibacterium acnes* overgrowth triggers the pathways that result in inflammatory acne lesions. 5α-reductase plays an active role in the local effects of androgens. The heterogeneity of 5α-reductase enzymes (isoenzymes types 1 and 2) explains the varying dermatologic effects of androgens.[22] Isoenzyme type 1 is present in sebaceous glands; type 2 is found in hair follicles. The clinical presentation of women with hyperandrogenism varies depending on the activity of these 2 isoenzymes. Serum levels of androgens do not seem to correlate with degree of hirsutism or acne—the sensitivity of androgen receptors and local levels of androgens play a more significant role.[30] This explains why many women with hirsutism and/or acne will not have an underlying endocrinopathy.

Alopecia is another important clinical feature of hyperandrogenism. Androgens stimulate the conversion of terminal follicles to vellus hair and also decrease the percentage of anagen hairs. This is achieved with local elevation of 5α-reductase levels and androgen receptors along with a decrease in

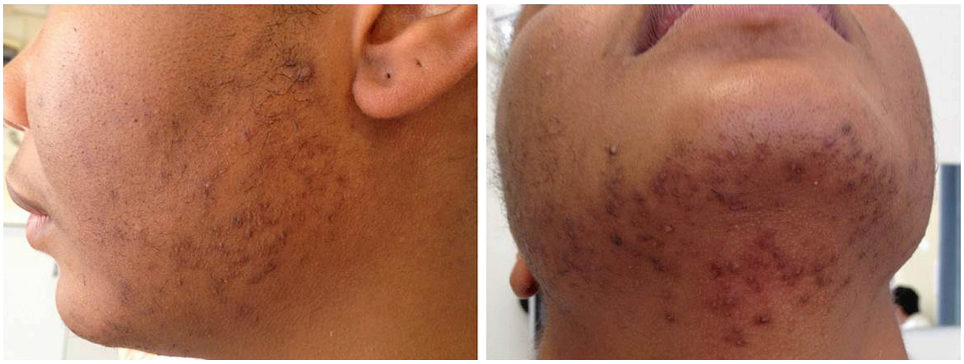

Fig 1. Hirsutism and acne are common dermatologic manifestations of polycystic ovary syndrome.

cytochrome P450, which reduces the conversion of testosterone to estrogen.[22,31] Among women with PCOS, alopecia is an infrequent finding.[32] For this reason, it is important to exclude other common causes of hair loss in women, such as thyroid abnormalities, iron deficiency anemia, alopecia areata, and telogen effluvium. Alopecia among women with PCOS can present with either a typical female pattern, with hair loss predominantly located in the central scalp with preservation of the frontal hairline, or, less commonly, male pattern baldness, with both frontotemporal and vertex recession.[32,33]

Chronic anovulation and endometrial cancer

PCOS is the leading cause of anovulatory infertility. Chronic anovulation can have pervasive consequences on fertility and oncologic risk. Women present with oligomenorrhea (<9 menses a year) or amenorrhea (missed menses for ≥3 months). These aberrant menstrual cycles often appear around the time of menarche, although they can occasionally appear later on in the setting of weight gain. Obese patients with PCOS who lose weight tend to have restoration of their menstrual cycles.[16,18] The ovaries are stimulated preferentially by LH, which results in ovarian androgen production. Local effects of androgens on the ovary arrest follicular development, preventing ovulation and progression into the luteal phase. In this context, estrogen levels are elevated without cyclical progesterone secretion. Progesterone is necessary to inhibit the proliferation and differentiation of the secretory endometrium. This constant stimulation of the endometrium by estrogen, unopposed by progesterone, increases the risk of endometrial hyperplasia and endometrial adenocarcinoma.[34] Other features associated with PCOS, such as hyperinsulinemia, elevated insulin-like growth factor (IGF-1), obesity, and hyperandrogenism also have mitogenic effects on the endometrium and are independently associated with endometrial cancer.[35]

Polycystic ovaries

According to the 2003 Rotterdam criteria, polycystic ovaries are 1 of the 3 diagnostic criteria. Polycystic ovaries on ultrasound are defined by the presence of ≥12 follicles in each ovary (each follicle measuring 2-9 mm in diameter) and/or an increased ovarian volume of >10 mL (Fig 2). When evaluating adolescent girls, ovarian volume size should be the sole criteria used to evaluate for polycystic ovaries because a transabdominal route is preferred and is less sensitive for the identification of follicles.[36] Among the general population of women with regular menstrual cycles and without any criteria for PCOS, 16% to 25% have polycystic ovaries on ultrasound.[37,38] Polycystic ovaries are found in 92% of women with hirsutism, 87% of women with oligomenorrhea, and 82% of reproductive age women with type 2 diabetes mellitus.[39,40] Insulin resistance, hyperandrogenism, and changes in SHBG seem to be involved in the development of the polycystic ovarian morphology, even among patients with ovulatory menstrual cycles.[41]

Other clinical features

Metabolic complications. PCOS is associated with several metabolic complications, most prominently metabolic syndrome, obesity, and insulin resistance. Up to 47% of women with PCOS have metabolic syndrome.[42] The diagnostic criteria for metabolic syndrome have been established by the Adult Treatment Panel (ATP) III and include ≥3 of the following: waist circumference >88 cm, triglyceride level ≥150 mg/dL, high-density lipoprotein cholesterol <50 mg/dL, blood pressure ≥130/85, and fasting glucose level ≥100 mg/dL. This close association between PCOS and metabolic syndrome appears to have an even stronger

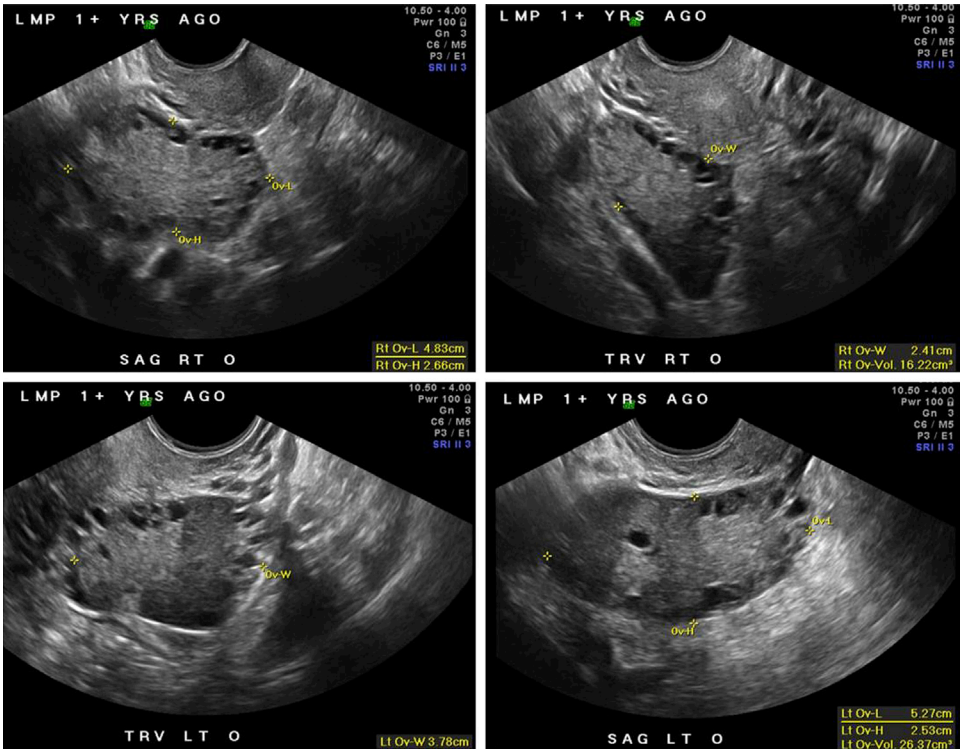

Fig 2. A transvaginal pelvic ultrasound scan reveals bilaterally enlarged ovaries with multiple small follicles of similar size along the periphery, which has a "string of pearls" appearance. The top 2 images show the right ovary (volume, 15 cc) in the sagittal and transverse views. The bottom 2 images show the left ovary (volume, 26 cc) in the sagittal and transverse views.

correlation among black women. Recently, a retrospective cohort study of 519 adolescents and adults with PCOS found a 44% increased risk for metabolic syndrome and cardiovascular disease among black women relative to white women.[43]

Obesity is present in as many as 75% of women with PCOS,[44] although this number varies depending on geography and is lower in other countries that have an overall lower prevalence of obesity, such as Finland, Greece, and Spain.[45-47] Central obesity appears to play a direct role in the pathophysiology of PCOS by contributing to insulin resistance and increasing androgen levels. In turn, hyperandrogenism and insulin resistance contribute to obesity, which perpetuates the cycle. A metaanalysis comparing obese to overweight women with PCOS revealed that obese women had significantly increased androgen, estrogen, and insulin levels compared to overweight women, further emphasizing the role that obesity has in the clinical picture of PCOS.[48]

Insulin resistance is common among patients with PCOS independent of obesity.[49] In a prospective controlled trial of 254 women with PCOS, 31% were found to have impaired glucose tolerance, while 7.5% had type 2 diabetes mellitus.[50] When examining the nonobese population with PCOS in that same study, the prevalence of impaired glucose tolerance was 10%, while the prevalence of diabetes was 1.5%.[50] While hyperinsulinemia is not part of the diagnostic criteria for PCOS, insulin plays an essential role in the development of anovulation and hyperandrogenism both by stimulating the theca cells to secrete testosterone and by decreasing the hepatic release of SHBG. This is further supported by the ability of insulin-sensitizing agents, such as metformin and thiazolidinedione, to lower androgen levels and induce ovulation.[51,52]

The cutaneous signs of hyperinsulinemia include acanthosis nigricans, striae distensae, and acrochordons. Acanthosis nigricans manifests as velvety hyperpigmented thickened plaques predominantly on the nape and sides of the neck, axillae, and groin. Elevated insulin levels bind to IGF-1 receptors, thereby stimulating proliferation of the epidermal keratinocytes and dermal fibroblasts.[53] Up to 50% of obese patients with PCOS have acanthosis nigricans, prompting an evaluation for impaired glucose tolerance in this population.[54]

Cardiovascular disease. Women with PCOS have an increased risk of developing cardiovascular disease. However, it is unclear if PCOS is an

independent risk factor for cardiovascular disease or a result of the comorbidities associated with PCOS, such as hypertension,[55] diabetes,[55] and dyslipidemia.[56,57] Patients with PCOS do have increased serum concentrations of cardiovascular disease risk markers, such as C-reactive protein, homocysteine, vascular endothelial growth factor, and plasminogen activator inibitor-1.[58] A study examining carotid intima media thickness as a surrogate for coronary artery disease found that patients with PCOS had a larger plaque index, even after controlling for body mass index (BMI), cholesterol level, and blood pressure.[59] In addition, coronary artery calcification, when measured via electron beam computed tomography, is more prevalent among women with PCOS.[60] A metaanalysis assessing the risk of coronary heart disease and stroke among patients with PCOS found a 2-fold risk compared to patients without PCOS. When adjusting for BMI, the risk increased by 55%.[61] Despite the compelling evidence, a prospective long-term study comparing women with PCOS to controls over a 20-year period concluded that postmenopausal women with PCOS do not have an increased number of cardiovascular events, despite having a strong cardiovascular risk profile.[62] However, this Scandinavian population had a smaller waist-to-hip ratio and lower BMI compared with other PCOS populations, which may not be representative of the group as a whole. While the data are controversial as to whether or not PCOS is an independent predictor of cardiovascular events, the consensus is that this population is vulnerable for cardiovascular disease and should be a target for primary prevention. Patients should be closely monitored and managed for obesity, diabetes, hyperlipidemia, and hypertension. The initial evaluation involves a fasting lipid panel, annual blood pressure and BMI calculations, and a 2-hour oral glucose tolerance test every 1 to 2 years.[63]

Obstructive sleep apnea, nonalcoholic steatohepatitis, and psychiatric manifestations. Patients with PCOS have a 5- to 10-fold higher risk of obstructive sleep apnea (OSA) compared to similarly obese patients without PCOS.[64] The etiology is in part hormonally mediated. Women with PCOS have decreased levels of progesterone, a hormone that has a protective effect by dilating upper airway muscles and decreasing airway resistance. Conversely, the elevated levels of testosterone among this population increase the apneic threshold. The combination of decreased progesterone levels and increased testosterone levels make the patient with PCOS susceptible for the development of OSA. Interestingly, treatment of OSA with continuous positive airway pressure has been shown to increase insulin sensitivity and decrease diastolic blood pressure, independent of changes in weight and fat distribution.[65] The physiology is thought to be driven by reductions in both norepinephrine levels and cardiac sympathetic activity.[58,66]

PCOS is also independently associated with nonalcoholic steatohepatitis. A study comparing women with PCOS to matched controls found that 44% compared to 20% had histologic nonalcoholic steatohepatitis after a liver biopsy specimen had been obtained, even after controlling for diabetes, obesity, and age.[67]

The psychological impact of obesity, infertility, hirsutism, and acne among women with PCOS is a source of recent interest. Women with PCOS have higher rates of depression, anxiety, and eating disorders.[68,69] Approximately 10% of women with PCOS suffer from these psychological conditions.[68] Changes in physical appearance, such as hirsutism and obesity, seem to play the greatest role in the psychosocial manifestations.[70]

In conclusion, PCOS is an increasingly common endocrinopathy. The pathophysiology represents a network of interconnecting hormonal pathways with the net result of elevated levels of androgens, insulin, and LH. These aberrant hormones have long-term metabolic, cardiovascular, oncologic, and reproductive implications. Dermatologists should be aware of the clinical features of PCOS and watch for the dermatologic findings of hyperandrogenism and insulin resistance, because these can often be the presenting manifestations of PCOS. Dermatologists are in a key position to make an early diagnosis of the syndrome and to ensure that the overall health risks of these patients are addressed.

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